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Abstract

Objective. To test whether personality traits were prospectively associated with type 2 diabetes incidence. **Methods.** The sample (*n* = 6798) was derived from the National Health and Nutrition Examination Survey Epidemiological Follow-up Study cohort. We fit four logistic regression models to test whether neuroticism, extraversion, openness to experience, or the Type A behavior pattern predicted type 2 diabetes incidence. Model 1 included sex, age, and race/ethnicity. Model 2 added personality traits, Model 3 added depressive symptoms, and Model 4 added body mass index (BMI), hypertension, and cigarette smoking status as predictors. **Results.** In Model 1 age was associated with increased risk of diabetes (2% per year); being black as opposed to white was associated with a three-fold increase in risk. In Model 2 age and being black were still significant and extraversion was associated with decreased risk (17% per standard deviation [SD]). In Model 3 age, being black, and extraversion were still significant. In addition, neuroticism was associated with decreased risk (26% per SD) and depressive symptoms were associated with increased risk (28% per SD). In Model 4 age, being black, neuroticism, and depressive symptoms were still significant. BMI was associated with increased risk (14% per SD) and each SD of depressive symptoms and extraversion was no longer significant. **Conclusions.** Higher neuroticism was associated with reduced type 2 diabetes risk even after controlling for race/ethnicity, age, depressive symptoms, and BMI. Extraversion and Type A behavior were not significant after including covariates.

Keywords: type 2 diabetes, incidence, personality, health, neuroticism, depression

Introduction

Diabetes mellitus is one of the biggest disease burdens of the modern age [1, 2]. The International Diabetes Federation lists obesity, unhealthy diet, physical inactivity, older age, insulin resistance, family history, and ethnicity as risk factors for type 2 diabetes [3]. There is also evidence that clinical variables such as depression and anxiety, as well as normal personality traits contribute to diabetes development.

A prospective study found that depressive symptoms were a risk factor for diabetes incidence in participants from the First National Health and Nutrition Examination Survey (NHANES I) Epidemiological Follow-up Study (NHEFS) [4]. A study of a large community sample found that the presence of depression increases type 2 diabetes risk by 6.87%, and that non-severe depression, persistent depression, and untreated depression are all associated with increased risk [5]. Similarly, two meta-analyses found that depressed adults are at greater risk of developing diabetes [6, 7]. Eriksson and colleagues [8] showed that baseline psychological distress, a measure of anxiety, depression, apathy, fatigue, and insomnia, predicted type 2 diabetes onset after controlling for baseline glucose tolerance. In a large prospective study by Engum [9], participants with higher scores on a baseline clinical measure that assessed anxiety and depression had significantly higher type 2 diabetes prevalence in the follow-up wave. Further studies suggest that symptoms of depression and anxiety influence type 2 diabetes risk by impairing endocrine response [10-12].

Normal personality traits have also been associated with diabetes. Two cross-sectional studies have shown that personality traits, such as those of the Five-Factor Model [13, 14], are associated with diabetes risk. In the first study, Goodwin and Friedman [15] found lower levels of conscientiousness and openness, and higher levels of agreeableness in people with diabetes than in people without diabetes. In the second, Goodwin, Cox, and Clara [16] reported that neuroticism was associated with increased risk of diabetes. On the other hand,

an individual participant meta-analysis of five cohort studies found that diabetes incidence was only associated with low levels of conscientiousness [17].

Studies have also shown associations between personality and physiological and behavioral factors related to diabetes risk. Neuroticism has been associated with the metabolic syndrome and its components, namely obesity, high triglycerides, hypertension, and elevated blood glucose [18, 19]. Tsenkova et al. [20] found that higher neuroticism enhanced the association between body mass index (BMI) and poor glucose metabolism. However, this same study also showed that neuroticism was *inversely* associated with average levels of blood sugar as measured by HbA_{1c}. Lower levels of neuroticism have been associated with unhealthy dietary habits [21] and lower levels of physical activity [22]. Higher extraversion has been associated with healthier dietary habits [21] and higher levels of physical activity [22]. Lower openness to experience has been associated with higher baseline cholesterol and tryglycerides levels [19] and poorer diet [21]. Finally, agreeableness has been associated with increased alcohol consumption [23, 24].

Given the findings of these studies we hypothesized that higher neuroticism would be associated with higher type 2 diabetes risk. We also hypothesized that higher openness would be associated with lower type 2 diabetes risk. Similarly, we hypothesized that, due to its association with low agreeableness [25], higher Type A behavior will be associated with higher type 2 diabetes risk. Finally, we hypothesized that extraversion would be associated with lower type 2 diabetes risk. We tested whether the effects of personality traits were attenuated by BMI, hypertension, smoking history and depressive symptoms.

Method

Participants

The sample was drawn from the NHEFS cohort [26, 27], which is a subsample of the NHANES I. The NHANES I was conducted in 1975 and was designed to assess the health

and nutritional status of a nationwide probability sample of approximately 32,000 participants in the United States [28, 29]. The NHEFS cohort comprises participants aged between 25 and 74 and took place over three follow-up waves (1982-1984, 1987, and 1992) [26, 27].

Our sample was comprised of men and women who completed the first (1982-1984) and final (1992) waves of assessment. The 1982 sample comprised 12,220 participants of which 3280 were excluded because of missing data on personality measures, depressive symptoms, diabetes, demographics, or medical covariates. The follow up 1992 sample consisted of 9281 participants. Of these, 753 were not classified with respect to the diabetes variables and were excluded. Merging the usable 1982 and 1992 subsamples produced a prospective sample with 6960 participants with full data at both time points. We further excluded participants if they reported having type 1 diabetes in 1982 and type 2 diabetes in 1992 (n = 6), if they had any type of diabetes in 1982 (n = 127), or if they had type 1 diabetes in 1992 (n = 29). The final sample thus comprised 6798 participants.

In 1982 the mean age of the final sample was 53.8 (SD = 13.8). The sample comprised 2429 men whose mean age in 1982 was 54.9 (SD = 13.9) and 4369 women whose mean age in 1982 was 53.2 (SD = 13.7). Of these participants, 6071 reported being “white”, 662 reported being “black”, and 65 reported being “other”. The last category included participants who reported themselves as Aleut, Eskimo, American Indian, Asian/Pacific islander, Hispanic, Japanese, Chinese, Korean, or Hindu.

One hundred and ninety seven (2.9%) participants developed type 2 diabetes over the ten year follow-up period. The incidence rates for females and males were 2.7% and 3.2%, respectively. These incidence rates are comparable to those reported during the same time period (3% incidence for women and 3.6% for men) [30].

Measures

Demographics. Age was treated as a continuous variable. Gender was coded 0 for females and 1 for males. Race/ethnicity was entered as two dummy-coded variables, which compared participants who self-identified as "black" or "other" to those who identified as "white", respectively.

Diabetes. Participants were classified as having developed type 2 diabetes based on their answers to two questions. The first asked: "Did a doctor ever tell you that you had diabetes or sugar diabetes?". The second question asked: "Are you *now* taking medications for this condition: Insulins (includes NPH U-100, Lente U-100, Lente Reg.)". Participants who answered "yes" to both questions were classified as having type 1 diabetes. Participants who answered "yes" to the first and "no" to the second question were classified as having type 2 diabetes. Participants who answered "no" to both questions were classified as not having diabetes.

Psychological variables. Personality and depressive symptoms were assessed in 1982. Short scales were used to assess neuroticism, extraversion, and openness to experience [31-33]. The Framingham Type A scale [34] was used to assess the Type A behavior pattern. The Center for Epidemiologic Studies Depression Scale [CES-D; 35] was used to assess depressive symptoms.

The neuroticism short scale consisted of five items chosen on theoretical grounds from the NHANES General Well-Being Schedule (GWBS) [36, 37]. Three neuroticism items were taken from the anxiety subscale of the GWBS. These items assessed the degree to which participants, during the past month, felt they were under strain, stress, or pressure, the degree to which participants were anxious, worried, or upset, and the degree to which participants were emotionally stable and sure of themselves (reverse scored) [36, 37]. The fourth neuroticism item was taken from the depression subscale of the GWBS and asked participants how cheerful/depressed they had been during the past month. The fifth

neuroticism item asked the participants how relaxed/tense they had been during the past month. The three items taken from the anxiety subscale were rated on a six point scale with lower scores indicating higher levels of endorsement. The two remaining items were rated from 0 to 10 with higher scores indicating higher levels of endorsement.

The extraversion and openness scales were selected from the NEO Inventory using multiple regression [32]. The eight (two reversed) extraversion items tapped aspects of extraversion related to gregariousness, warmth, activity, assertiveness, positive emotions, and excitement-seeking. The six (three reversed) openness items tapped aspects of openness related to openness to fantasy, actions, aesthetics, ideas. The extraversion and openness items could be rated on a five point scale ranging from 1 “Strongly Disagree” to 5 “Strongly Agree”. We scored these scales according to their scoring keys [31].

The internal consistencies of the neuroticism, extraversion, and openness scales were identified in a previous study as being .76, .51 and .42, respectively [33]. The lower internal consistencies for the latter two scales were attributed to “the breadth of item content as well as the brevity of the scales” [33, p. 142]. In addition to internal consistencies, these scales showed good convergent and discriminant validity against self, spouse, and peer reports on the NEO Inventory’s neuroticism, extraversion, and openness scales [32]. The neuroticism and extraversion scales also displayed good convergent and discriminant validity against the neuroticism and extraversion scales of the Eysenck Personality Inventory [38].

The fourth personality measure was the Framingham Type A scale [34]. This scale is designed to assess a stable behavioral pattern characterized by competitiveness, striving for achievement, aggressiveness, and time urgency. The scale has been shown to be related to the measures of hostility and the low pole of the agreeableness domain of the Five-Factor Model [25, 39, 40]. For 6613 participants, the scale consisted of six items that could be answered using a four-point Likert scale with higher scores indicating less of the trait. A small subset of

participants (n = 158) were presented with a three point Likert scale. We treated these participants as if none of them endorsed the missing point. The internal consistency in the analysed sample was .58. Omitting these participants from the main analyses did not change the results.

Depressive symptoms were assessed using the Center for Epidemiologic Studies Depression Scale [CES-D; 35], which was designed to measure depressive symptomatology in the general population. It consists of twenty items to assess symptoms of depression such as feeling sad, lonely, having crying spells, feeling happy (reverse-scored). The answers are given on a 4-point scale that assesses the frequency of reported symptoms, ranging from "Rarely of none of the time" to "Most or all of the time". We used a continuous measure of depression rather than a cut-point to parallel the continuous personality traits.

Health. While weight data were available in the NHEFS cohort, height data were not. We thus used height data from the NHANES I and weight data from the NHEFS to compute BMI. The correlation between weight in the NHANES I and first wave of NHEFS was high, $r(6796) = .88$, $p < .001$. We treated BMI as a continuous variable.

Hypertension status was based on participants' answer to the question "Has a doctor ever prescribed medication for you for hypertension or high blood pressure?" Responses were coded 0 for "no" and 1 for "yes".

Smoking status was based on participants' answers to two questions: "Have you ever smoked more than 100 cigarettes?" and "Are you a smoker now?" Participants answering "yes" to both questions were classified as current smokers. Participants answering "yes" to the former and "no" to the latter were classified as former smokers. Participants answering "no" to both questions were classified as non-smokers. Smoking status was entered as two dummy coded variables: the first compared former smokers to non-smokers and the second compared current smokers to non-smokers.

Analyses

Descriptive statistics. We used *t*-tests and chi-square tests to compare participants who developed type 2 diabetes during the 10 years of follow-up to those who did not develop type 2 diabetes. Similarly, we compared participants included versus those excluded from the final sample due to missingness or not meeting the study selection criteria.

Logistic regression models. To assess personality risk factors for type 2 diabetes incidence, we fit generalized linear models using the glm function in R, version 3.0.3 [41]. A set of models assessed personality risk factors for type 2 diabetes incidence. We were interested in whether potential associations between neuroticism, extraversion, openness, and Type A were mediated through depressive symptoms, or traditional risk factors such as BMI, hypertension, and smoking history. We fit four models. Model 1 tested for the effects of gender, race/ethnicity, and age on development of type 2 diabetes. Model 2 included the same predictors as in Model 1 and the four personality traits. Model 3 tested whether the effects of personality traits, controlling for demographics, would be attenuated by including depression in the model. Finally, Model 4 added risk factors such as smoking, hypertension, and BMI, to test for further attenuation of the effects of personality. For ease of interpretation, psychological variables were transformed into *z*-scores.

Results

Table 1 presents descriptive statistics for the whole sample and by type 2 diabetes status. Table 2 presents the differences in means and frequencies between participants in the initial sample and those included in the study, and between those who did and did not develop diabetes. The differences between those included and excluded from the study even though significant were small in size, and ranged between just under one tenth of a standard deviation for Type A behavior to just over one third of a standard deviation for depressive

symptoms. Bivariate correlations of the key variables at baseline for the participants included in the analyses are presented in Table 3.

The logistic regression models are presented in Table 4. The linearity assumption was met for all variables in all of the models. Across all models, the variance inflation factor [42] ranged from 1.01 to 1.89. There was thus little evidence that multicollinearity was a problem as would be indicated if the variance inflation factor were greater than five.

In Model 1, gender was not significantly related to type 2 diabetes risk. However, age was a significant risk factor, with a 2% increase in risk per year of age and race/ethnicity was also a risk factor, with participants categorized as black being approximately three times as likely to report developing type 2 diabetes.

Model 2 included the effects of personality. In this model, the effect sizes for the demographic predictors were similar to those in Model 1. Extraversion was significantly related to type 2 diabetes incidence with each standard deviation increase of extraversion being associated with a 17% reduction in risk.

Model 3 included the effects of depression. In this model, depression was a significant risk factor, with each standard deviation being associated with a 28% increase in risk. The effect of neuroticism was now significant with each standard deviation increase being related to a 26% reduction in risk for type 2 diabetes. Extraversion was still significant with each standard deviation related to a 15% reduction in risk.

Finally, Model 4 included BMI, hypertension, and smoking. The effects of BMI but not smoking or hypertension, was a significant risk factor: each unit increase in BMI was associated with 14% increase in risk. Increased neuroticism was still associated with a 25% reduction in risk per standard deviation. However, the effect of depressive symptoms was somewhat attenuated, with each standard deviation now being associated with 23% increase in risk. Other personality traits were not significantly related to diabetes risk.

The neuroticism and depressive symptoms variables were positively skewed. To correct this we square root transformed both variables. We then re-ran Models 3 and 4. After the transformation, the effect size of neuroticism dropped from 26% to 20% in Model 3 and from 25% to 20% in Model 4. The neuroticism effects remained significant in both models. After transformation, the effect size for depressive symptoms dropped from 28% to 21% in Model 3 and from 23% to 17% in Model 4. The effects of depressive symptoms effects were still significant in Model 3, but no longer significant in Model 4 (p = .086).

Discussion

In a model controlling for age and race/ethnicity, higher extraversion was associated with reduced risk for developing type 2 diabetes. After adjusting for depressive symptoms, extraversion was still associated with reduced risk. Moreover, higher neuroticism was now significantly associated with reduced risk. After also adjusting for BMI, hypertension, and smoking, neuroticism was still significant and protective factor, extraversion was no longer significantly related to risk of type 2 diabetes.

Finding that *lower* neuroticism is related to diabetes risk is surprising given studies indicating that, along with depressive symptoms, anxiety is linked to higher diabetes risk [8, 9], particularly as both chronic anxiety and depression are associated with neuroticism [43]. To try and resolve these differences we first considered several methodological reasons that could have led to our result. In light of the fact that including depressive symptoms in the model brought out the protective effect of neuroticism, and that neuroticism and depression are phenotypically and genetically related [43-46], these findings may have resulted from overfitting. However, this is not a plausible explanation for three reasons: 1) unadjusted mean levels of neuroticism were higher in the group that did not go on to develop diabetes; 2) the direction of the neuroticism effect was the same even in a model that did not include

depressive symptoms; 3) the variance inflation factor values ranged from 1.01 to 1.89 and thus were far below a cut-point of 5 that would suggest multicollinearity.

Another possibility is that the protective effect of neuroticism came about because of attrition. Participants who were included in the study were significantly more neurotic and depressed than those who were excluded. However, it is difficult to see how this difference would reverse the effect of neuroticism, but not that of depressive symptoms. Furthermore, the differences in means for the included and excluded samples were, while significant, very small – approximately one fifth of a standard deviation for neuroticism, and one third of a standard deviation for depressive symptoms. Finally, the nature and magnitude of the correlation between the neuroticism and depressive symptoms did not differ between the total NHEFS sample (.64), the sample used in this study (.62), and in participants who were excluded from the study (.68). Thus, attrition did not have an effect on the association between neuroticism and a well-known criterion. It is therefore unlikely that the observed effects of neuroticism were due to attrition.

Having ruled out the possibility that overfitting or attrition could explain the present findings with respect to neuroticism, we turn to differences between our study and previous studies. Two of the previous studies [8, 9] that found positive associations between anxiety measures and diabetes risk used measures that combined symptoms of anxiety and depression into a single score. As such, these measures could simply tap depression to a greater extent than anxiety. Moreover, the elevated endocrine response that would result in increased risk of diabetes [10-12] may not be reflected in the normal range of stress and worry captured by trait neuroticism [47]. Thus, even though neuroticism is a risk factor for anxiety and depression [43], there may be a difference between the effect subclinical levels of depression and anxiety, as operationalized by neuroticism, and more serious symptoms of depression and anxiety have on health outcomes.

The present findings also differ from studies that used similar neuroticism measures because, while we used a prospective design, the studies that found a positive association between neuroticism and type 2 diabetes were cross-sectional [15, 16]. Thus, the findings in these other studies may reflect reverse causality.

The possibility that neuroticism is, in fact, protective, is supported by two recent findings in the diabetes literature. First, higher neuroticism was related to *better* glycaemic regulation in patients with type 2 diabetes [48]. Second, Tsenkova and her colleagues [20] found a positive association between neuroticism and immediate blood sugar markers, such as blood insulin, but a negative association between neuroticism and HbA_{1c}, a measure of blood sugar concentration over time. More broadly, Turiano et al. [49], showed that participants high in neuroticism who were also high in conscientiousness had lower circulating interleukin-6 levels than those with any other configuration of the two traits. Furthermore, a recent study showed that lower neuroticism is associated with shorter telomere length, a predictor of mortality and late life disease onset [50]. Similarly, studies show neuroticism to be associated with greater vigilance [51] and perceived susceptibility to health risks [52], which may lead to individuals seeking out physicians at pre-diabetic stages, when the condition can still be reversed.

The effect of Type A behavior was not significant and the effect of extraversion, although still significant after controlling for depressive symptoms, was not significant after controlling for BMI, suggesting that extraversion may influence diabetes risk through factors such as physical activity [22] and diet [21].

The present study had some limitations. First, the NHEFS cohort was not assessed on a measure of conscientiousness, a well-established personality predictor of health behaviors and outcomes [53, 54] or on constructs such as Type D personality [56]. Second, the personality scales did not allow us to examine these effects at the facet level [55]. Third,

diabetes status and hypertension were based on self-reports. However, prior studies indicate close correspondence between self-reports of medical conditions and actual disease diagnoses [57, 58]. Furthermore, self-reported diabetes in the NHEFS cohort has been shown to predict all-cause mortality [59]. Future studies should thus endeavor to include measures of insulin resistance, blood glucose levels, and HbA1c, as well as cholesterol and triglyceride levels, or should be based on actual type 2 diabetes diagnosis. They should also include broader measures of personality that would allow one to examine these associations at the facet level. These steps would allow for more accurate assessment of the associations between personality, traditional risk factors, and type 2 diabetes. Fourth, even though the food and alcohol intake and physical activity variables were available, the number of participants with missing data on these variables was high and thus including these variables would greatly reduce the available sample size. However, we used BMI as a proxy of healthy diet and physical activity [60, 61]. Finally, we were unable to control for family history of diabetes. Future studies should therefore control for family history, possibly in genetically-informative designs.

The present study had several strengths. First, the sample was large and representative. Second, the study included several covariates related to diabetes risk. Third, the detailed and repeated interview data enabled us to screen out probable cases of type 1 diabetes and individuals who were diagnosed as having diabetes at or before baseline.

While it is tempting to suggest, based on these findings, that patients who are high in neuroticism should be screened for diabetes less often than patients who are low in neuroticism, this is overly simplistic, and could very well be harmful. Instead, these results suggest that it may be worth considering different prevention strategies for patients who have high and low levels of neuroticism. On the one hand, patients who are high in neuroticism should be screened for depressive symptoms as they are clear risk factors for diabetes. On the

other hand, patients who are low in neuroticism should be taught to be vigilant for the signs of high blood sugar or diabetes risk factors.

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Table 1

Descriptive Statistics of the Final Sample by 1992 Diabetes Status

	Type 2 Diabetes		
	Absent	Present	Total
	(*n* = 6601)	(*n* = 197)	(*n* = 6798)
Variable	*M (SD)*	*M (SD)*	*M (SD)*
Neuroticism	9.7 (6.5)	8.8 (6.4)	9.6 (6.5)
Extraversion	18.3 (3.6)	17.7 (3.5)	18.3 (3.6)
Openness	12.0 (3.0)	11.6 (3.0)	12.0 (3.0)
Type A	13.4 (3.6)	13.5 (3.7)	13.4 (3.6)
Depression	7.7 (7.7)	8.9 (8.4)	7.7 (7.8)
BMI (kg/m^2)	25.9 (4.6)	29.8 (5.5)	26.0 (4.7)
Age (at baseline)	53.7 (13.8)	57.4 (12.9)	53.8 (13.8)
	n (%)	*n* (%)	*n* (%)
Gender			
Male	2352 (35.6)	77 (39.1)	2429 (35.7)
Female	4249 (64.4)	120 (60.9)	4369 (64.3)
Race/Ethnicity			
White	5923 (89.7)	148 (75.1)	6071 (89.3)
Black	616 (9.3)	46 (23.4)	662 (9.7)
Other	62 (1.0)	3 (1.5)	65 (1.0)
Hypertension			
Present	1680 (25.5)	86 (43.7)	1766 (26.0)
Absent	4921 (74.5)	111 (56.3)	5032 (74.0)

	Type 2 Diabetes		
	Absent	Present	Total
	(*n* = 6601)	(*n* = 197)	(*n* = 6798)
Smoking status			
Non-smoker	2985 (45.2)	85 (43.1)	3070 (45.1)
Former smoker	1741 (26.4)	55 (27.9)	1796 (26.4)
Current smoker	1875 (28.4)	57 (29.0)	1932 (28.5)

Note. Personality and depression scores are given in raw units.

Table 2

Differences in means and frequencies for all variables.

	Type 2 vs. None			Excluded vs. Included		
	t	*df*	*p*	*t*	*df*	*p*
Neuroticism	1.92	6796	.054	-8.25	9950	< .001
Extraversion	2.36	6796	.018	9.52	9888	< .001
Openness	1.44	6796	.15	10.25	9989	< .001
Type A	-0.34	6796	.73	4.04	9809	< .001
Depression	-2.05	6796	.040	-14.97	9510	< .001
BMI (kg/m^2)	-11.68	6796	< .001	-8.48	9970	< .001
	χ^2	*df*	*p*	χ^2	*df*	*p*
Gender	0.85	1	.36	140.72	1	< .001
Race/Ethnicity	43.85	2	< .001	225.03	2	< .001
Hypertension	32.03	1	< .001	314.19	1	< .001
Smoking	0.37	2	.83	110.45	2	< .001

Note. Equal variances assumed for *t*-tests.

Table 3

Pearson's correlations between main variables at baseline

	Neuroticism	CES-D	Extraversion	Openness	Type A	BMI
Age	-.16***	.03*	-.16***	-.17***	-.26***	.01
Neuroticism		.62***	-.10***	.04***	.26***	-.02
CES-D			-.15***	-.03*	.16***	.03*
Extraversion				.23***	.18***	-.01
Openness					.06***	-.02
Type A						.06***

Note. CES-D = The Center for Epidemiologic Studies Depression Scale; n = 6798;
*** $p < .001$, ** $p < .01$, * $p < .05$

Table 4

Odds Ratios and 95% Confidence Intervals of Type 2 Diabetes Incidence Associated with Demographics, Personality Traits, Depression, and Diabetes-related Factors

	Model 1		Model 2		Model 3		Model 4	
	OR (95% *CI*)	*p*	*OR* (95% *CI*)	*p*	*OR* (95% *CI*)	*p*	*OR* (95% *CI*)	*p*
Female vs. Male	1.16 (0.87-1.56)	.31	1.08 (0.79-1.46)	.64	1.11 (0.82-1.50)	.51	1.19 (0.86-1.66)	.30
Race/Ethnicity								
White vs. Black	3.03 (2.15-4.27)	< .001	3.02 (2.14-4.25)	< .001	2.83 (2.00-4.00)	< .001	1.99 (1.38-2.87)	< .001
White vs. Other	2.03 (0.63-6.55)	.24	1.99 (0.61-6.43)	.25	1.94 (0.60-6.28)	.27	2.42 (0.74-7.95)	.15
Age	1.02 (1.01-1.03)	< .001	1.02 (1.01-1.03)	< .001	1.02 (1.01-1.03)	.003	1.02 (1.01-1.03)	.002
Neuroticism			0.86 (0.74-1.01)	.064	0.74 (0.61-0.90)	.002	0.75 (0.61-0.91)	.004
Openness to Experience			0.99 (0.85-1.15)	.85	0.99 (0.85-1.15)	.88	0.98 (0.84-1.14)	.79
Extraversion			0.83 (0.72-0.97)	.019	0.85 (0.73-.99)	.038	0.87 (0.75-1.01)	.073
Type A			1.16 (1.00-1.36)	.053	1.14 (0.98-1.33)	.085	1.09 (0.93-1.27)	.30
Depression					1.28 (1.07-1.52)	.006	1.23 (1.02-1.47)	.026
BMI							1.14 (1.11-1.17)	< .001

	Model 1		Model 2		Model 3		Model 4	
	OR (95% *CI*)	*p*	*OR* (95% *CI*)	*p*	*OR* (95% *CI*)	*p*	*OR* (95% *CI*)	*p*
Hypertension							1.29 (0.94-1.79)	.12
Smoking status								
Non vs. Former							1.12 (0.77-1.63)	.54
Non vs. Current							1.40 (0.97-2.03)	.070

Note. Personality predictors in terms of the effects of standard deviation increases. *n* = 6798 for all models.